Cardiorespiratory Function

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PULMONARY FUNCTION has been evaluated on a basis of the ability of the lungs to supply oxygen to and remove carbon dioxide from the blood during rest and exercise. In the large majority of cases of pulmonary disease with dyspnea the disability has been found due to fibrosis and emphysema. So great is the pulmonary reserve in man that clinical or roentgenologic detection of those conditions at an early stage is difficult. Physiologic tests of pulmonary function in addition to the clinical and roentgenological examination have been found necessary to appraise lung function accurately in the presence of fibrosis and emphysema. Physiological tests have been studied and analyzed with respect to the early detection of susceptible individuals to environmental hazards before serious stages of disability develop, for the early recognition and removal of such persons would greatly benefit both employees and industry.

The basic data for this discussion were obtained from a study of 500 miners of soft and hard coal who had fibrosis and emphysema and respiratory complaints. Physiological studies on such a large homogeneous group supply data from which a satisfactory evaluation can be made of the significance and limitations of the various physiological tests employed in pulmonary function measurements in man.

The measurements made were: Maximal breathing capacity[5] and vital capacity from spirogram tracings employing special apparatus (before and after treatment with a bronchodilator drug); residual air volume by the oxygen open circuit method;[1] arterial blood oxygen saturation both at rest and immediately after step-up exercise by the Van Slyke content and capacity method;[21] direct arterial pO_2 and pCO_2 determinations, by the Riley bubble method;[17] arterial pH at rest and after exercise; oxygen uptake and carbon dioxide output during rest and exercise from direct gas analysis of inspired and expired air;[6] per cent of oxygen extraction by the lungs from inspired air both during rest and exercise; and the duration of dyspnea after one minute of step-up exercise (30 steps on a stool 20 cm. high, in one minute).

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• Accurate evaluation of the degree of impairment of pulmonary function in persons with fibrosis and emphysema requires: (1) ventilatory measurements from rapid spirogram tracings (vital capacity, maximal breathing capacity and the time required to blow the air from the lungs); (2) determination of the degree of bronchospasm present; (3) determination of the degree of pulmonary emphysema (residual air expressed quantitatively as percent of total lung volume); (4) determination of the arterial blood oxygen saturation at rest and immediately after step-up exercise; (5) measurement of the oxygen extraction from inspired air (per cent of oxygen removed) during rest and exercise; (6) determination of the oxygen up-take during exercise; and (7) observation of the duration of dyspnea after step-up exercise.

No single physiologic test is adequate in evaluating impairment of pulmonary function, and roentgenograms of the chest are unreliable as a sole basis for appraisal of disability.

In industrial medicine, pulmonary function studies make possible (a) more accurate diagnosis and evaluation of pulmonary disability; and (b) earlier detection and thus prevention of prolonged exposure by susceptible individuals to environmental hazards.

Spirogram tracings of vital capacity and maximal breathing capacity, residual air volume measurements and exercise tolerance tests are the most important standardized tests of pulmonary function for clinical detection of fibrosis and emphysema. Bronchospasm plays a prominent role in chronic pulmonary disease and this aspect needs to be determined. Determination of arterial blood oxygen saturations and the percentage of oxygen extracted from the air breathed are frequently necessary for accurate evaluation. A mild exercise test is necessary, but not one designed as a measure of physical fitness. Spirogram tracings of ventilation measurements, which are easy to obtain with suitable apparatus, are most useful in screening tests, for abnormalities may be detected by this means long before roentgenologic changes appear.

On a basis of the physiological studies the 500 coal miners were classified into four groups as regards degree of impairment of pulmonary function —slight, moderate, advanced and far advanced.

A complete history, physical examination and radiological study supply important information for the clinical evaluation of impairment. However, such information frequently proves inadequate for an accurate determination of the extent of disability present and in some cases even fails to indicate the nature of the respiratory difficulty. In recent years the discrepancy between roentgenological studies and pulmonary function studies, in the appraisal of disability produced by silicosis in coal miners, has become well recognized in many parts of the world.[2, 3, 7, 8]

In the subjects of this study there was no apparent correlation between the roentgenographic stage of silicosis and the degree of impairment of pulmonary function (Table 1). Third stage silicosis existed in cases in which there was a slight degree of pulmonary function impairment, and advanced degrees of function impairment occurred in cases in which silicosis was only borderline or in the first stage. A lack of correlation of the roentgenographic stage of silicosis and the degree of pulmonary emphysema has been found in both hard[7] and soft[8] coal miners. Roentgenograms of the chest, although useful in diagnosing silicosis, should not be used as the only criteria in disability evaluation. The disease cannot be diagnosed on the basis of pulmonary function measurements alone, but the information available from these studies provides for more exact appraisal of the disability present than is possible by the usual clinical diagnostic procedures.

Pulmonary emphysema and chronic bronchitis are commonly regarded as the two factors producing disability in coal miner's silicosis. The presence and extent of pulmonary emphysema was accurately determined (duplicate checks of 100 cc. or less in all cases) in this study by measuring the residual air volume, using an improved oxygen open circuit method[1, 6] with a demand valve. The residual air volume was expressed as a percentage of the total lung volume (residual air plus vital capacity). This value expresses as a physiological relationship the effect of an increased volume of residual air on the mixing and dilution factor with respect to vital capacity and total lung volume. Thus it is immaterial whether the total lung volume is increased, decreased or remains unchanged, for the percentage value obtained represents the functional value of the pulmonary emphysema. The air remaining in the lungs after a forced expiration is termed residual air, and this normally occupies about 25 per cent of total lung volume.

The 500 cases in this study were classified according to the degree of pulmonary emphysema based

TABLE 1.—*Degree of Impairment of Pulmonary Function as Compared with Stage of Silicosis (Roentgenographically Determined) in 500 Coal Miners with Respiratory Complaints*

Degree of Impairment of Pulmonary Function	Group	Cases Studied No.	Cases Studied Per Cent	Roentgenologic Classification* Normal No.	Normal Per Cent	Borderline No.	Borderline Per Cent	First No.	First Per Cent	Second No.	Second Per Cent	Third No.	Third Per Cent
Slight	1	82	16.4	1	1.23	10	12.20	15	18.29	27	32.92	29	35.36
Moderate	2	169	33.8	7	4.14	15	8.88	23	13.61	35	20.71	89	52.66
Advanced	3	170	34.0	3	1.77	10	5.88	20	11.76	23	13.53	114	67.06
Far advanced	4	79	15.8	0	0.00	2	2.53	13	16.46	17	21.52	47	59.49
Total		500	100.0	11	2.20	37	7.40	71	14.20	102	20.40	279	55.80

* *Borderline*, increased peribronchial markings; *first stage*, slight fine mottling in parenchyma and increased size and density of hilar lymph nodes; *second stage*, typical nodular round and oblong shadows of soft even density from 2 to 6 mm. in diameter; *third stage*, shadows larger than 6 mm. in diameter and coalescence into aggregates.

TABLE 2.—*Degree of Pulmonary Emphysema (Data based on the residual air percentage of total lung volume in 500 coal miners with respiratory complaints)*

Degree of Emphysema	Classification by Groups	Residual Percentage of Total Lung Volume	Significant Clinically	Cases Studied No.	Cases Studied Pct.	Roentgenologic Classification* Normal No.	Normal Pct.	Borderline No.	Borderline Pct.	First No.	First Pct.	Second No.	Second Pct.	Third No.	Third Pct.
None	1	25 or less	No	38	7.6	1	9.09	6	16.22	3	4.23	13	12.75	15	5.38
Slight	2	25-35	No†	138	27.6	3	27.27	8	21.62	18	25.35	34	33.33	75	26.88
Moderate	3	35-45	Yes	165	33.0	6	54.55	13	35.13	17	23.94	23	22.55	106	37.99
Advanced	4	45-55	Yes	106	21.2	1	9.09	7	18.92	19	26.76	19	18.62	60	21.51
Far advanced	5	Above 55	Yes	53	10.6	0	0.00	3	8.11	14	19.72	13	12.75	23	8.24
Total				500	100.0	11	....	37	....	71	....	102	....	279	....

* See footnote, Table 1.

† If other function measurements are normal, hyperventilation compensates for the slight increase in the residual air volume.

on the residual air percentage of total lung volume (Table 2). Although this percentage grouping is somewhat arbitrary, other related physiologic measurements substantiate the validity of the classification for diagnosis, study and evaluation.[6] Thus in 35 per cent of the 500 cases, pulmonary emphysema was not a significant factor in producing disability (Table 2). Residual air measurements were not obtained during periods of upper respiratory infections or asthmatic attacks or other temporary abnormalities which were clinically apparent, so that the residual air measurement represents the best baseline measurement obtainable in each case. In a comparison study of the residual air volumes at rest and during a steady state of moderate treadmill exercise, in 51 cases the changes in residual air percentage of total lung volume were found to be small (average increase 1.4 per cent) even in extensive pulmonary emphysema.[9] Upper respiratory infections or asthmatic attacks produce temporary decreases in vital capacity and increases in residual air, so that during the involved period, the mixing and dilution problem is increased in some cases to pronounced degree. An increased residual air volume represents a potential hazard which may produce complete incapacity during periods of respiratory infections. A residual air volume over 35 per cent of total lung volume is abnormal and indicates the presence of emphysema in clinically significant degree. In some cases a marked degree of reversible bronchospasm exists. In this condition treatment with bronchodilator drugs results in some reduction of residual air and some increase of vital capacity, but the normal state is not restored and the change in residual air percentage of total lung volume is usually small if the baseline study is obtained as described.

A high degree of reversible bronchospasm may exist without an increased volume of residual air. Some patients with a considerable increase in residual air volume have minimal disability, as compensatory hyperventilation is good, and the dyspnea level is not reached even with moderate exercise. This indicates the value of good ventilation (vital capacity and maximal breathing capacity) in limiting the disability resulting from pulmonary emphysema. A correlation was noted between the degree of emphysema and the incidence of strain upon the right side of the heart,[4] but the typical electrocardiographic findings are absent in about 40 per cent of the cases with a far advanced degree of emphysema (over 55 per cent of total lung volume residual air). Emphysema of a far advanced degree is disabling and the evaluation of disability may be made on this measurement alone. The residual air volume determination is one of the most important function measurements in disability appraisal. An increased residual air volume (above 35 per cent of total lung volume) is abnormal and represents emphysema, not a temporary insufflation of the lungs.

Vital capacity, although a useful and essential measurement, has but limited value alone. In general, vital capacity decreases as the degree of pulmonary emphysema increases[10] but because of the wide range of individual variations the usefulness of this measurement is limited, unless the values are extremely low (less than 1,800 cc. in men) or very high (above 4,200 cc. in men). A vital capacity of 2,000 to 3,000 cc. may exist without emphysema or with a far advanced degree of emphysema, with slight disability or total disability. The shape of the spirogram tracing[7] supplies useful information on the state of the lungs, as well as giving the vital capacity volume. When more than three seconds is required to blow most of the air out of the lungs after a deep inspiration, impairment is indicated. This may be due to loss of elasticity or to obstruction or to both. The time element and the shape of the spirogram tracing should be taken into consideration in analyzing vital capacity tracings (Figure 1). This time factor is usually ignored. The three-second vital capacity represents the maximal portion that is of use, for if a longer time interval is required, the individual cannot make use of the added portion with breathing rates of 12 to 15 per minute.

Maximal breathing capacity is both a time and volume measurement, and the normal values for men range from 120 to 180 liters per minute, depending on the age and size of the subjects and the technique used.[5] In general, if the maximal breathing capacity (MBC) measurement is less than 40 liters per minute in men the degree of pulmonary emphysema and function impairment is significant, but insignificant if the MBC is over 120 liters per minute, and otherwise indeterminate for values between 40 and 120 liters per minute. If the MBC is good, hyperventilation can compensate partially or completely for the mixing and dilution factor imposed by the increased residual air volume in pulmonary emphysema. In general, compensatory hyperventilation is adequate in the absence of other complicating factors for increases in residual air volumes up to about 35 per cent of total lung volume[11] as shown by changes (see Figure 2) in arterial pCO_2 (increased above 40 mm. of mercury). Above this value, however, hyperventilation is often inadequate to maintain normal gas exchange.

Since measurement of the maximal breathing capacity appears to have practical value in the early detection of fibrosis and bronchial obstruction it would seem desirable to take MBC measurements routinely along with chest roentgenograms of industrial workers exposed to dust hazards. If the MBC

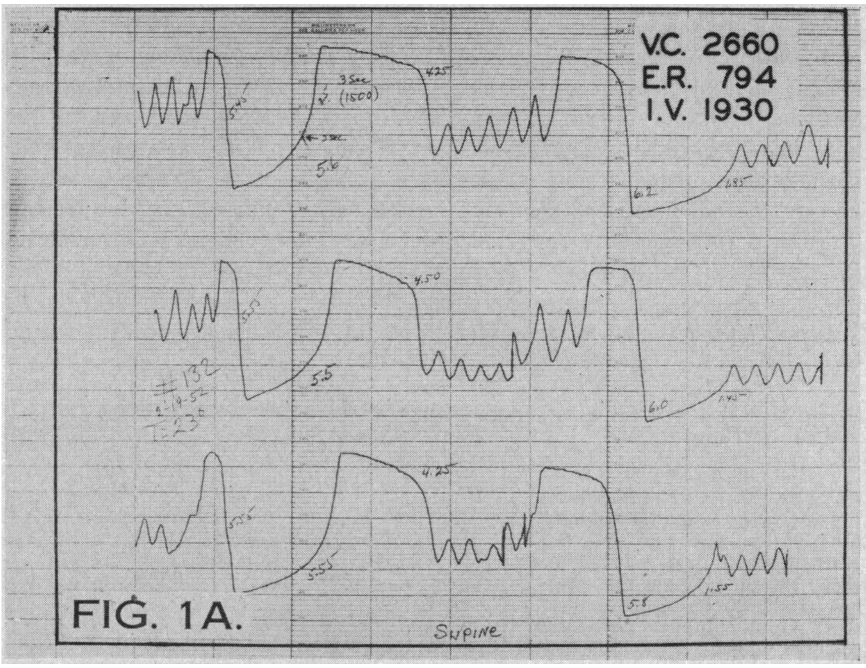

Figure 1-A.—Spirogram tracings of vital capacity. Read from right to left. Time intervals between vertical lines, 12 seconds. Vital capacity (VC) was measured both by having the subject take the deepest possible breath after a forced expiration and exhale as far as possible after taking the deepest possible breath. In chronic pulmonary disease one may give a slightly higher figure than the other, but in normal persons there is no significant difference. Note the abnormal shape of the spirogram tracing and the prolonged time required for exhalation after a deep inspiration. The normal exhalation time should be three seconds or less. The expiratory reserve (ER) is the part below the quiet breathing baseline, and the inspiratory volume (IV) is the part above this baseline. The volume of air exhaled in the first three seconds represents the maximal usable part of the VC measurement. The rest of the volume of air shown is nonfunctional.

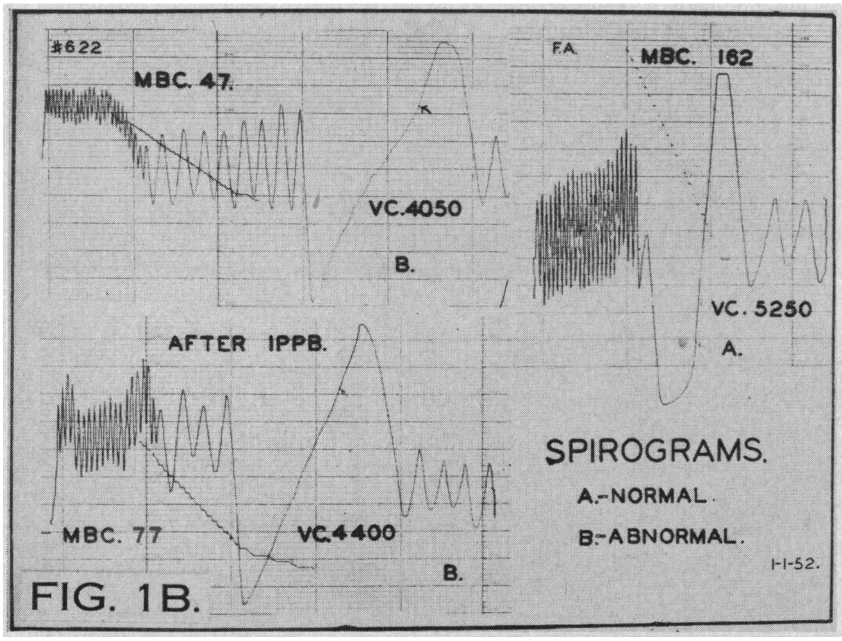

Figure 1-B.—Normal and abnormal spirogram tracings of vital capacity and maximal breathing capacity are shown. Note in the abnormal tracing 22 seconds was required to complete exhalation and although the total vital capacity volume was 4,050 cc., actually the maximal usable part of vital capacity was 852 cc. (that volume exhaled during the first three seconds). The lower part of the abnormal tracing was after intermittent positive pressure breathing (PPB) treatment with a bronchodilator drug, Vaponefrin.® Note the abnormal shape of the spirogram tracing is still present although the maximal breathing capacity (MBC) was increased from 47 to 77 liters per minute.

measurements decrease progressively from year to year, the indication is that fibrosis and loss of lung elasticity with obstruction are developing and the individual should be removed from dust hazards, regardless of roentgenologic appearance of the chest at the time.

The arterial blood oxygen saturation was determined routinely in all cases both at rest (basal type) and immediately after step-up exercise, and in addition in 122 cases during a steady state of treadmill exercise. A comparison of the arterial blood oxygen saturation after exercise with the saturation when the subject is at rest is most important in disability evaluation. If the saturation drops 5 per cent or more with exercise the prognosis is poor; whereas, if the saturation increases above the resting value with exercise the prognosis is favorable. The resting arterial blood oxygen saturation was poorly correlated with pulmonary function impairment.[6] A normal resting arterial blood oxygen saturation (96 per cent) has been observed to decrease 10 per cent with mild step-up exercise. Some persons who have subnormal oxygen concentration in arterial blood while at rest (even as low as 88 per cent) have increases of from 5 to 8 per cent upon exercise, a change brought about by the more uniform alveolar aeration accompanying the deeper breathing.[12]

Direct measurements of arterial pO_2 (Figure 2) provide a more sensitive index of early changes impairing the respiratory gas exchange than the arterial blood oxygen saturation (as determined from the content and capacity measurements), because the magnitude of the pO_2 changes is much greater and this greater range permits a better analysis of the specific factors producing impairment in the respiratory gas exchange. The effective inspired pO_2 varies with the barometric pressure and the proportion of oxygen in the inspired air. The average pO_2 sea level values of breathing air are: inspired, 150 mm. (mercury); alveolar, 100 mm.; arterial blood,

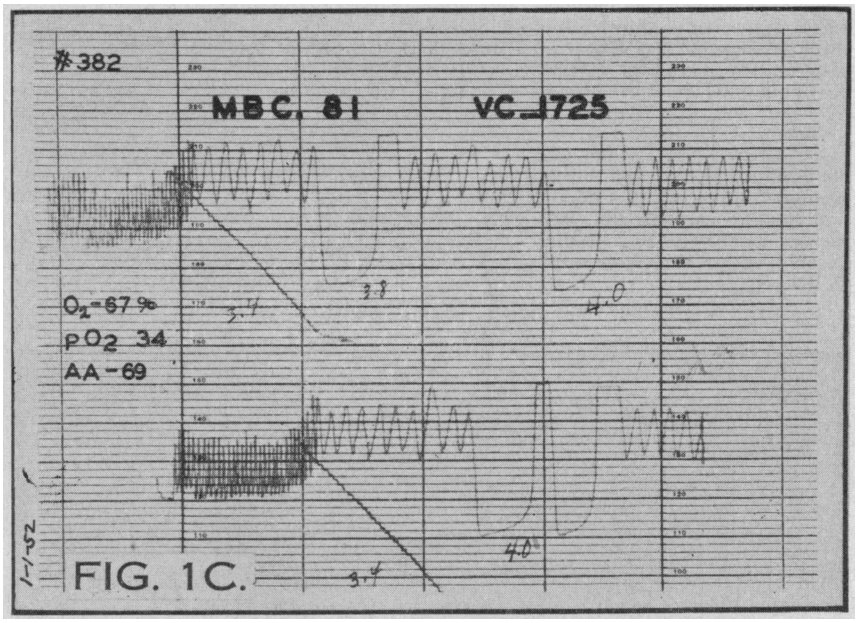

Figure 1-C.—A spirogram tracing of a very low vital capacity of 1,725 cc., but note that the entire volume was exhaled in three seconds; hence the effective vital capacity in this patient was actually higher than the 4,050 cc. shown in the abnormal tracing in Figure 1-B. The maximal breathing capacity was 81 liters per minute, indicative of a rapid exhalation. This tracing demonstrates the importance of recording the shape of the exhalation pattern by a rapid spirogram tracing, and that the three-second vital capacity is a much more significant measurement than the total vital capacity volume when the time factor is ignored.

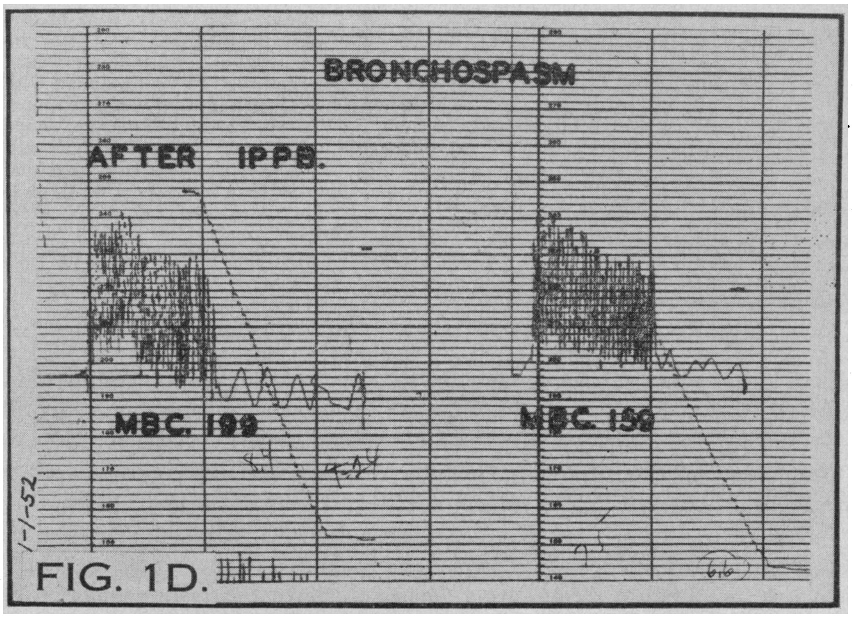

Figure 1-D.—Spirogram tracings of maximal breathing capacity in a normal person with a value of 159 liters before and 199 liters immediately after one treatment with intermittent positive pressure breathing and Vaponefrin.® Diagnosis: Chronic tracheobronchitis with bronchospasm secondary to postnasal discharge of chronic sinusitis.

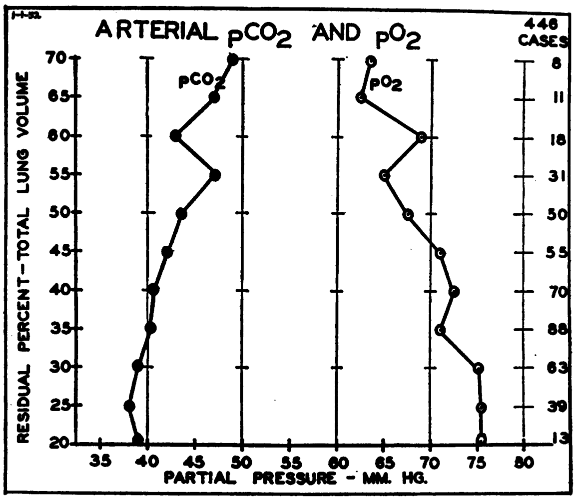

Figure 2.—Correlation between the residual percentage of total lung volume on the ordinate and the arterial pCO_2 and pO_2 partial pressures, in mm. of mercury, by direct determination in 446 cases of silicosis with fibrosis and emphysema. The individual points plotted for pCO_2 and pO_2 represent the average for the number of cases in each range of 5 per cent for the residual per cent of total lung volume (for example, 88 cases are represented with an average residual per cent of total lung volume of 35 per cent, plus or minus 2.5 per cent). It can be noted that when the residual percentage of total lung volume was greater than 35 per cent, the arterial pCO_2 was greater than 40 mm. of mercury, and this was increased progressively as the residual percentage of total lung volume was increased. The arterial pO_2 was decreased in the lowest group (residual 20 per cent of total lung volume) to 75 mm. of mercury (normal 100 mm. of mercury). The average pO_2 values were decreased progressively as the residual percentage of total lung volume was increased. A good correlation was found to exist between the increased arterial pCO_2 and the decreased arterial pO_2 with the increased residual percentage of total lung volume.

95 mm. If the expired air is collected at the same time as the arterial blood sample, the mean alveolar pO_2 can be calculated by the indirect method[18] (Figure 3) which assumes that the alveolar pCO_2 is the same as the arterial pCO_2. The mean alveolar pO_2 has been checked and reduplicated consistently by the indirect method in all types of patients with chronic pulmonary disease. Calculated arterial pO_2 values were found unreliable in studying patients with chronic pulmonary disease. In an analysis of 446 cases (Figure 3) the alveolar-arterial pO_2 difference was uniformly elevated independent of the residual per cent of total lung volume, but the alveolar pO_2 and inspired-alveolar pO_2 difference were correlated with the residual volume changes.

The distribution factor is the aspect most commonly involved, producing interference with the alveolar-arterial gas exchange in chronic pulmonary disease (Figure 4). Distribution refers to unequal alveolar aeration and perfusion, and the aeration factor concerns alveolar ventilation. At sea level the alveolar mean pO_2 is normally about 100 mm. of mercury, and blood perfusing alveoli will take up oxygen to a pO_2 level of about 95 mm. of mercury (normally saturated 96 per cent).

Alveolar aeration is impaired by loss of elasticity, fibrosis, obstruction from mucous plugs, secretions, partial atelectasis or depressed respiratory movement, as all of these conditions restrict the free movement of air in and out of the alveoli. Impaired alveolar aeration results in a lowering of the partial pressure of oxygen in the alveoli involved. Blood perfusing poorly aerated alveoli is incompletely saturated, but flows to the left side of the heart to mix with the normally saturated blood. The perfusion aspect of the distribution factor also includes those alveoli with complete aeration blockage but in which the blood supply is maintained. Such an alteration produces little shunts, there being no gas exchange in the blocked alveoli. Other alveoli may be ventilated but not perfused because the capillary blood supply is obliterated by fibrosis, thrombosis or vasoconstriction. The aerated but non-perfused alveoli represent physiologic dead space and decrease lung ventilation efficiency. The distribution factor has been found to be independent of the degree of pulmonary emphysema[6] (Figure 3).

The elevated mean alveolar-arterial oxygen gradient was decreased and the arterial oxygen saturation increased by intermittent positive pressure breathing using compressed air,[16] a response independent of the hyperventilation and increased blowing off of CO_2 produced by the respirators. The evidence from the intermittent positive pressure breathing data suggests that a more uniform alveolar aeration is accomplished by better inflation of those alveoli with impaired air circulation[13] (see Figure 4), partially correcting the interference produced by the mechanical obstruction resulting from fibrosis and the attendant loss of elasticity, often accentuated by bronchospasm. Some alveoli may be opened during intermittent positive pressure breathing which were totally closed during ambient breathing.

The per cent of oxygen removed from the air breathed in is a measure of the lung ventilation efficiency (normal 4 to 5 per cent at rest and 5 to 6 per cent during exercise, at sea level). For example, if the amount of oxygen removed from the air breathed is 4.5 per cent, then 45 cc. of oxygen is extracted from each liter of air breathed. If the amount of oxygen removed from the air breathed were greatly reduced, the volume of inspired air required would be proportionately increased for a given amount of work and oxygen consumption; hence, if the ventilatory measurements (MBC and vital capacity) are re-

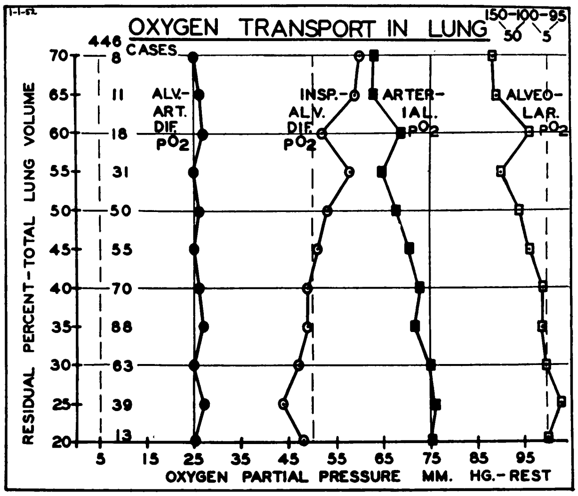

Figure 3.—The oxygen transport in the lungs was analyzed in the 446 cases referred to in Figure 2, using the same case grouping based on the residual per cent of total lung volume on the ordinate. The alveolar pO_2 was decreased progressively as the residual per cent of total lung volume was increased. If the residual was 40 per cent or more of total lung volume, the alveolar pO_2 was less than the normal value of 100 mm. of mercury. The arterial pO_2 values are parallel to the alveolar pO_2 changes. The difference between the inspired pO_2 and alveolar pO_2 was plotted in the same manner and correlated with the residual per cent of total lung volume. When the residual was 40 per cent or more of total lung volume, the inspired-alveolar pO_2 difference was greater than the normal sea level value of 50 mm. of mercury. The observed values less than 50 mm. of mercury resulted from hyperventilation when the residual was 35 per cent or less of total lung volume. An increased residual volume increases the difference between the inspired and alveolar pO_2, lowers the alveolar pO_2 and lowers the arterial pO_2 and the per cent saturation of the blood. When the alveolar-arterial pO_2 difference was plotted with the residual per cent of total lung volume there was no correlation. The average alveolar-arterial pO_2 difference was about 25 mm. of mercury, and this occurred in all of the groups, indicating the factor responsible for this gradient to be independent of the residual per cent of total lung volume. The alveolar-arterial pO_2 difference above was due to the distribution factor which is shown more graphically in Figure 4.

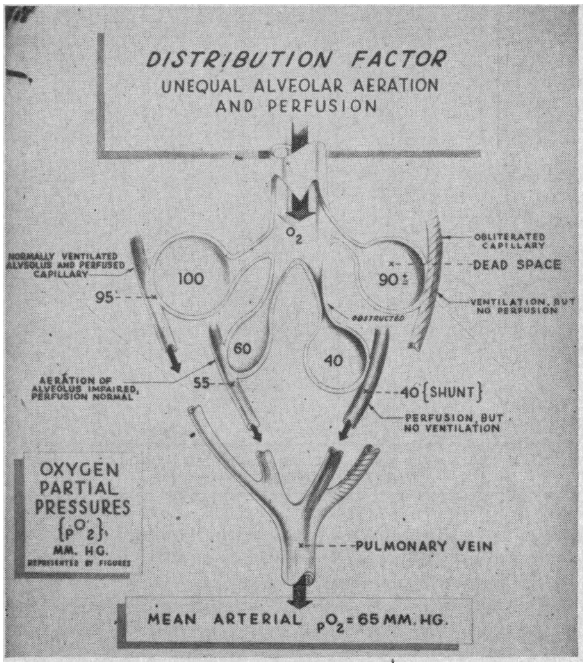

Figure 4.—A schematic diagram indicating the disturbed relationship between alveolar aeration and perfusion, as produced by pulmonary fibrosis, retained secretions, bronchospasm, emphysema, consolidation, pulmonary atelectasis or depression of respiration. Intermittent positive pressure breathing increases the effective pO_2 in alveoli with impaired aeration, but still perfused with blood, and increases the per cent saturation of the arterial blood. Intermittent positive pressure breathing has no effect on the oxygen transfer in alveoli which are perfused but non-aerated unless some alveoli are opened up by the increased inspiratory pressure. The distribution factor is characteristic of chronic pulmonary disease with fibrosis and emphysema.

duced, the individual may be unable to meet the elevated oxygen requirement. No correlation was found between the degree of pulmonary emphysema and the per cent of oxygen removed at rest (Figure 5), and only a slight average decrease with exercise. In most instances the lung ventilation efficiency was reduced, but this test as an isolated measurement was of limited value in estimating disability.[14]

The duration of dyspnea was determined subjectively after the step-up exercise test (30 steps on a stool 20 cm. high in one minute). This test is a practical one for pulmonary function, although not as a measure of physical fitness. Normal duration of dyspnea after the test is 90 seconds. Duration of dyspnea up to 120 seconds is a *slight* prolongation, from 120 to 180 seconds a *moderate* prolongation, and upward of 180 seconds *advanced* prolongation. When the ventilatory, residual air and arterial blood saturation measurements are essentially normal, prolonged dyspnea after the step-up test indicates increased pulmonary vascular resistance. A significant reduction in the expansibility of the pulmonary bed is indicated when the oxygen uptake is less than 400 cc. per square meter of body surface area during the one minute step-up exercise. Increased pulmonary vascular resistance especially during exercise is a factor in most cases of chronic fibrosis and emphysema, and is the primary factor in some cases as evidenced by the profound dyspnea after the one minute step-up test. Since the duration of dyspnea is a subjective test, the significance of a prolonged time requires careful evaluation in the individual case and is subject to the limitations of this type of test.

No correlation was noted between the degree of pulmonary emphysema and the circulation times obtained by either the arm to lung (ether) or arm to tongue (Decholin®) tests[14] (Figure 6). A pronounced

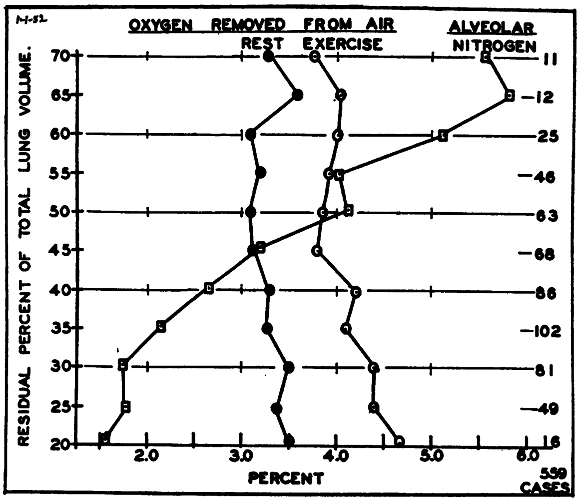

Figure 5.—The correlation between the per cent of oxygen removed from the inspired air both during rest and exercise and the residual per cent of total lung volume for 559 cases (as in Figure 2). The per cent of oxygen removed from the inspired air was decreased slightly at rest and during exercise as the residual per cent of total lung volume was increased. In contrast, note the good correlation obtained by plotting the alveolar nitrogen per cent after breathing 100 per cent oxygen for seven minutes with the residual per cent of total lung volume. The per cent of oxygen removed from the inspired air during rest and exercise (may be expressed as a ventilatory equivalent) was reduced some from the normal in most cases, but unless this value was very low it was not a significant factor in evaluating disability or pulmonary function.

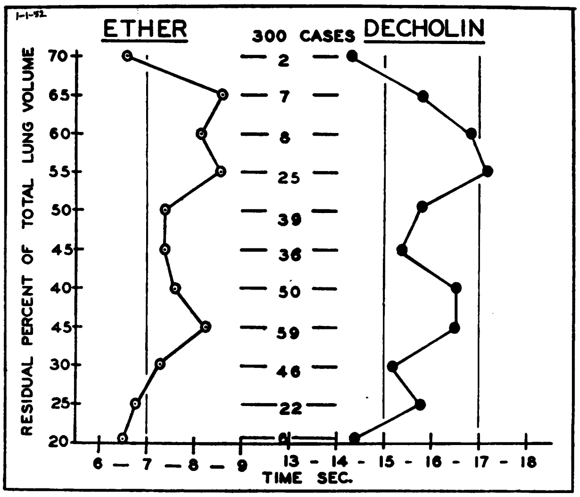

Figure 6.—The ether (arm to lung) and Decholin® (arm to tongue) circulation times correlated with the residual per cent of total lung volume in 300 unselected cases of fibrosis and emphysema. The number of cases for each group is shown in the center. There was no significant difference for either the ether or the Decholin circulation times with respect to the degree of emphysema as indicated by the residual per cent of total lung volume. If either the ether or the Decholin time were abnormally prolonged, a definite cardiac factor was indicated.

prolongation of circulation time indicated definite cardiac involvement.

Plasma volume studies in 138 coal miners with silicosis and respiratory complaints (using the Gregerson T-1824 dye method) revealed an increase of 10.2 per cent in the emphysema group and 5.4 per cent in the non-emphysema group[20] with an average packed cell volume of 42.4 per cent of the whole blood. The plasma volume was decreased slightly during treadmill exercise.

The average oxygen capacity in 115 cases of soft coal miners and 385 cases of hard coal miners was 19.48 volumes per cent (equivalent to 14.54 gm. of hemoglobin) with a standard deviation of 1.267 volumes per cent (0.95 gm. hemoglobin). There was no difference in the two groups of miners. Polycythemia is not a factor in coal miner's silicosis, even in cases of pronounced cyanosis and emphysema, in the absence of frank cardiac failure. An elevated hemoglobin content indicates cardiac failure, but not fibrosis and emphysema *per se*.

There was no apparent correlation between pulse and respiration rates during rest and exercise and the degree of impairment of pulmonary function[14] because of the wide standard deviation observed in each group.[14]

Dyspnea[19] is the most common complaint of coal miners with silicosis, a symptom resulting from fibrosis and/or emphysema. However, only 65 per cent of all coal miners studied had a significant degree of pulmonary emphysema (Table 2). Dyspnea commonly results from alteration in breathing resistance produced by increase in the air-flow velocity. This may accompany exertion or any other situation which actively increases pulmonary ventilation such as the acute hypoxia which may occur with lipiodol instillation.[15] Bronchial obstruction is an important factor producing increased breathing resistance in many cases. Bronchial obstruction[16] may be either permanently fixed or reversible in various degrees by inhalation of bronchodilator drugs. The reversible type is referred to as bronchospasm and the relief afforded by the bronchodilator drug is probably mostly from the relief of the bronchospasm, although the vasoconstrictor element may be of some importance. Bronchospasm occurs in normal persons during infection of the upper respiratory tract, but this temporary abnormality does not hamper activity, owing to the large breathing reserve present. Fibrosis with loss of lung elasticity tends to cause retention of secretions, enhances irritation of the bronchial mucosa and predisposes to bronchospasm with defective aeration of alveoli, especially in the lung bases in cases in which there is pronounced impairment of diaphragmatic movement.

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REFERENCES

1. Darling, R. C., Cournand, A., and Richards, D. W., Jr.: Studies on intrapulmonary mixture of gases: an open circuit method for measuring residual air, J. Clin. Invest., 19:609, 1940.

2. Fletcher, C. M.: Pneumonoconiosis of coal miners, Brit. Med. J., 1:1015, 1948.

3. Gordon, B., Motley, H. L., Theodos, P. A., Lang, L. P.: Anthracosilicosis and its symptomatic treatment, West Virginia Med. J., 45:126, 1949.

4. Lang, L. P. and Motley, H. L.: Electrocardiographic findings in anthracosilicosis, Federation Proc., 8:91, 1949.

5. Motley, H. L., and Tomashefski, J. F.: Studies on maximal breathing capacity measurements, Am. J. Physiol., 163: 736, 1950.

6. Motley, H. L., Lang, L. P. and Gordon, B.: Studies in the respiratory gas exchange in one hundred anthracite miners with pulmonary complaints, Am. Rev. Tuberc., 61: 201, 1950.

7. Motley, H. L., Gordon, B., Lang, L. P. and Theodos, P. A.: Impairment of pulmonary function in anthracosilicosis, Arch. Ind. Hyg. and Occupational Med., 1:133, 1950.

8. Motley, H. L.: Pulmonary function studies in bituminous coal miners, West Virginia Med. J., 46:8, 1950.

9. Motley, H. L. and Tomashefski, J. F.: Studies of residual air volumes at rest and during treadmill exercise, Federation Proc., 10:94, 1951.

10. Motley, H. L., Lang, L. P. and Gordon, B.: Pulmonary emphysema and ventilation measurements in one hundred anthracite coal miners with respiratory complaints, Am. Rev. Tuberc., 59:270, 1949.

11. Motley, H. L.: Experimentation on the Effects of Respirators on the Respiratory Gas Exchange, Medical Division Report, Army Chemical Center, Maryland, No. 5, pp. 51-65, April 1951.

12. Motley, H. L. and Tomashefski, J. F.: Comparison of step-up and treadmill exercise using arterial blood and respiratory gas exchange measurements, Federation Proc., 9:92, 1950.

13. Motley, H. L. and Tomashefski, J. F.: Effect of high and low oxygen levels and intermittent positive pressure breathing on oxygen transport in the lungs in pulmonary fibrosis and emphysema, J. Applied Physiol., 3:189, 1950.

14. Motley, H. L.: Evaluation of pulmonary function impairment by physiological studies on 400 unselected cases of hard and soft coal miners with respiratory complaints, McIntyre Research Foundation Conference on Silicosis and Aluminum Therapy, Schumacker, Ont., pp. 49-67, Jan. 30, 1951.

15. Motley, H. L. and Tomashefski, J. F.: The acute effects of lipiodol instillation on the respiratory gas exchange, Am. J. Physiol., 167:812, 1951.

16. Motley, H. L. and Tomashefski, J. F.: The treatment of chronic pulmonary disease with intermittent positive pressure breathing. 1, The evaluation by objective physiological measurements, Arch. Ind. Hyg. and Occupational Med., 5:1, 1952.

17. Riley, R. L., Proemmel, D.D. and Franke, R. E.: A direct method for determination of oxygen and carbon dioxide tensions in blood, J. Biol. Chem., 161:121, 1945.

18. Riley, R. L., Lilienthal, J. L., Jr., Proemmel, D. D. and Franke, R. E.: On the determination of the physiologically effective pressure of oxygen and carbon dioxide in alveolar air, Am. J. Physiol., 147: 191, 1946.

19. Theodos, P. A., Gordon, B., Lang, L. P., and Motley, H. L.: Studies in disability in anthracosilicosis, Dis. Chest, 17:249, 1950.

20. Tomashefski, J. F. and Motley, H. L.: Plasma and blood volume studies at rest and during exercise in coal miners with fibrosis and emphysema, Am. J. Physiol., 167: 832, 1951.

21. Van Slyke, D. D. and Neill, G. M.: The determination of cases in blood and other solutions by vacuum extraction and manometric measurement, J. Biol. Chem., 61:523, 1924.

Funds for Medical Schools

THE SUM of $671,834 was turned over by the American Medical Education Foundation to the National Fund for Medical Education for distribution to the 79 medical schools in the United States. This represents the amount collected from physicians during the first six months of 1952. This money added to the amount collected from industry by the National Fund for Medical Education was distributed July 31 in the form of grants amounting to $15,000 to each of the 72 four-year schools and $7,500 to each of the seven two-year schools.

—*A.M.A. News Notes*